Ontario

ROYAL COMMISSION OF INQUIRY INTO CERTAIN DEATHS AT THE HOSPITAL FOR SICK CHILDREN AND RELATED MATTERS.

Hearing held
21st floor
180 Dundas Street West
Toronto, Ontario

The Honourable Mr. Justice S.G.M. Grange	Commissioner
P.S.A. Lamek, Q.C.	Counsel
E.A. Cronk	Associate Counsel
Thomas Millar	Administrator

Transcript of evidence
for

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Hearing held on the 21st Floor, 180 Dundas Street West, Toronto, Ontario, on Thursday, the 7th day of June 1984.

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THE HONOURABLE MR. JUSTICE S.G.M. GRANGE - Commissioner

THOMAS MILLAR - Administrator

MURRAY R. ELLIOT - Registrar

APPEARANCES:

P.S.A. LAMEK, Q.C. E. CRONK	Commission Counsel
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I.G. SCOTT, Q.C. M. THOMSON	Counsel for The Hospital for Sick Children
D. YOUNG	Counsel for The Metropolitan Toronto Police
W.N. ORTVED	Counsel for numerous Doctors at The Hospital for Sick Children
F. KITELY	Counsel for the Registered Nurses' Association of Ontario and 35 Registered Nurses at The Hospital for Sick Children

APPEARANCES: (Cont'd)

D. BROWN	)	Counsel for Susan Nelles - Nurse
G.R. STRATHY	)	Counsel for Phyllis Trayner - Nurse
J.A. OLAH	)	Counsel for Janet Brownless - R.N.A.
S. LABOW	)	Counsel for Mr. & Mrs. Gosselin, Mr. & Mrs. Gionas, Mr. & Mrs. Inwood, Mr. & Mrs. Turner, Mr. & Mrs. Lutes, and Mr. & Mrs. Murphy (parents of deceased children)
W.W. TOBIAS	)	Counsel for Mr. & Mrs. Hines (parents of deceased child Jordan Hines)
J. SHINEHOFT	)	Counsel for Lorie Pacsai and Kevin Garnet (parents of deceased child Kevin Pacsai)

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I N D E X

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---On commencing at 10:00 a.m.

THE COMMISSIONER: Yes, Mr. Lamek.

MR. LAMEK: Mr. Commissioner, before going on to the next child I want to go back and correct something that I said yesterday in the course of the submissions I was making about Justin Cook. With respect to the possibility of a drug error at midnight on the night that Cook died I said that he had been given .4 milligrams of Inderal and among the reasons that I advanced for rejecting that as an opportunity for an effective error, I said that if on the pharmacologists' evidence .6 milligrams at 3:50 in the morning would not be sufficient to produce the levels recorded if digoxin was substituted for that amount, then a fortiori if digoxin was substituted for the .4 milligrams four hours earlier at midnight, it could not have had that result. I am obliged to say to you that I misread the medication sheet in the chart in making that submission. On page 17 of the Cook chart and this is borne out by the psysician's orders on pages 14 and 15, it is clear I believe that the standing order for Inderal, that was the one that was given at midnight, was initially for 3 milligrams not .3 milligrams, and that was increased to 4 milligrams

not .4 milligrams. As stated therefore the point I was making was invalid.

It is, however, still my submission that an effective medications error at midnight is not plausible. At midnight Cook was given 3 milligrams of the oral Inderal preparation and 1 milligram, that is to say 1cc in volume of the parenteral variety.

In Dr. Kauffman's report, that is Exhibit 266, and the page to which I refer you, sir, is numbered variously 4 in typed script at the top in the centre and 308 in a numbering machine on the right hand side. On that page, the last two paragraphs on the page, Dr. Kauffman is attempting to estimate an oral dose of digoxin elixir which would produce the serum concentration recorded, and calculates that an oral dose of elixir containing .7 milligrams of digoxin, that is to say one which would be contained in about 14 millilitres or cubic centimetres of fluid, given one hour before death, would produce the recorded levels. He goes on if it were given six hours before death you need six milligrams of digoxin, which in the elixir would be contained in 120 ccs of fluid. Midnight then being four or five hours before death if that is reasonable to say that the oral dose required at that stage to produce the

levels perhaps come closer to the 6 milligrams than to the .7 milligram dose postulated by Dr. Kauffman and perhaps occupy a volume of fluid of 75 to 100 cubic centimetres.

Now in the final paragraph Dr. Kauffman says this:

"It is highly unlikely that the dose was administered orally since this would be extremely difficult considering the condition of the infant during the hours prior to death and the volume of paediatric elixir required to deliver the dose."

Meaning it is a lot more difficult to deliver the dose if it is crushed tablets.

Dr. Kauffman's view is clearly against the oral route of administration simply because of the volume of liquid that is required. It is apparent, although we don't know, to my knowledge we don't know the concentration of the oral preparation of Inderal, it is apparent I suggest that the concentration is very much higher than that of the oral preparation of digoxin. Otherwise a dose of 3 milligrams of the oral Inderal would have been a very healthy drink indeed for young Cook at midnight,

there is no suggestion of that.

At the end of the day, sir, it is still my submission that a midnight drug administration, an error, is not a likely time for medication error of the kind that is going to be effective in producing a level that was recorded in Justin Cook at 4:30 in the morning.

Therefore my submission with respect to Cook remains as I said it yesterday, that you are entitled to find, and indeed should find that the child died by digoxin intoxication resulting from a deliberately administered overdose of digoxin.

Now, if sir you should accept that submission as to how and by what means Justin Cook died, the consequences I suggest have to be very significant. As you have said on earlier occasions before the Court of Appeal Judgment on the naming of names, you can't have murder without a murderer.

I don't necessarily say that Justin Cook was murdered. I do say and the distinction although it is perhaps somewhat metaphysical for normal people, that is to say people other than lawyers, is nevertheless I think an important one. I do say that someone deliberately administered a large overdose of digoxin to Cook and thereby caused

his death.

You of course are not permitted to express any view as to who that person might be, and certainly I make no submissions going to that point.

The point I do make is not unlike the point you earlier made. If my submission as to the cause and means of Justin Cook's death be valid, it follows that there was somebody on the night of March 21 and 22, 1981 in the Hospital for Sick Children who had access to Justin Cook and who committed the deed of deliberate administration of an overdose. There was somebody abroad in the Hospital with the capacity to do that thing. And the knowledge that such a person existed necessarily adds a new circumstance to your consideration of this whole matter.

The notion which entirely understandably was unthinkable to the physicians and nurses, the notion that in a place of healing someone should be deliberately harming sick helpless infants, that notion not only ceases to be unthinkable from the moment you accept my submission about Cook, if you do, that notion becomes a fact, there was somebody who deliberately harmed and killed Justin Cook.

What was speculation becomes fact, and

the fact adds a new and frankly horrible dimension to the consideration not only of the other 35 deaths considered singly, but also the totality of the events on the cardiology wards in July of 1980 until March of 1981.

Justin Cook was not the only child for whom, in my submission you should find death resulted from a deliberately administered overdose, but no other case I suggest is quite so clear. There are several other children about whom I will suggest you may properly find either that their deaths were caused, or that there is a strong probability that their deaths were caused by a deliberately administered overdose of digoxin.

I propose next, Mr. Commissioner, to move successively to three groups of children. First the three other children with whose murder Miss Nelles was charged, that is to say babies Miller, Pacsai and Estrella. Then to the three children other than Justin Cook for whom digoxin had never been prescribed but in whose bodies it was found, babies Hines, Lombardo and Belanger. Then to a group of five children who were so desparately ill that no resuscitation effort was made when they suffered cardiac arrests, and those children are Alan Perreault,

Paul Murphy, Laurette Heyworth, Bruce Floryn and David Leith.

In the context of referring to that last group, sir, the do not resuscitate group, let me add this as a further legacy of an acceptance of my submission as to Justin Cook. If there was a killer at work on Wards 4A and 4B the fact that a child was tragically terminally ill, certain to die, beyond hope or help, is no necessary indication that child's death when it occurred was a natural one. No matter how consistent the child's death may have been with his clinical condition and course, no death can be ruled out of the suspicious category without a close examination of all of the circumstances to see if any features exist to raise suspicion.

Let me move then, sir, to the case of Allana Miller. That child was three days short of her first birthday when she died in the early morning hours of March the 21st. During the last six months of her life Dr. Freedom had seen her in office visits on a referral from a physician in Kitchener. Her course to the time of admission to the Hospital for Sick Children is neatly and concisely plotted in Dr. Freedom's reporting letters on pages 6, 8 and 10 of the chart.

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THE COMMISSIONER: Sorry, the page?

MR. LAMEK: Page 6 is the first of the reporting letters. That is dated October 24 of 1980. Page 8, letter of December 18, 1980. Page 10, a letter of March 4, 1981. There is a further letter at page 12 to which I will come in a moment, sir.

It was thought initially in October of 1980 as appears in a letter that begins on page 6 that surgical repair of Allana Miller's cardiac anomalies could be put off for a year or two. Dr. Freedom was on the alert for changes in her course that might indicate the need for earlier surgery.

She was sent home to be treated with digoxin and diuretics at home.

She was seen again by Dr. Freedom in December of 1980. He wrote his reporting letter the day after that visit on December 18. She was noted at that time to have mild congestive heart failure. She was mildly tachypneic and she was thought to be progressing satisfactorily.

There was still no decision to have any earlier surgery scheduled than had originally been contemplated, but Dr. Freedom remained alert for changes in the condition of the child.

He saw her again on March 3rd, 1981 and sent a reporting letter to the referring physician on March 4, 1981. It begins on page 10 of the chart. It appears that in the period since her December visit she has had several periods of hospitalization in Kitchener for chest infections and there had been a failure to thrive.

Now in fact back in October one of the matters that Dr. Freedom had said they needed to be aware of and look out for was repeated chest infections. Now that apparently was a situation that had developed. She was now seen to be not progressing satisfactorily. There was significant congestive heart failure which was not apparently responding satisfactorily to the digoxin and diuretics with which she had now been treated for six months, and it was proposed by Dr. Freedom to admit her within the next four to six weeks for surgery. It was apparently no longer feasible to wait the year or two they originally had in mind and hoped for. It was an apparently deteriorating condition which required some prompt action but not it would seem emergency action.

On page 12 of the chart there is a further letter written by Dr. Freedom in which he reports that Allana Miller's case had been discussed

at the medical surgical staff conference on the 9th of March, and everyone had agreed that the surgery should proceed sooner rather than later and the surgery had in fact been scheduled for later in March.

Indeed we note from the chart, sir, elsewhere that surgery was scheduled for March 29th, 1981, some two and a half weeks later. In other words there was now a decision to have earlier surgery than had been earlier hoped; earlier surgery than Dr. Freedom had contemplated even on March 4th, but we still do not seem to be dealing with an urgent critical situation of the kind that we saw in Justin Cook's case. Surgery was scheduled for Sunday morning two days after admission.

Allana Miller was admitted to the Hospital, to ward 4A, on March 19th late in the evening. Her brief course in the Hospital was characterized as appears from the nursing note, pages 41 and 42, by irregularity and slowness of heart rate, and need to be in 40% oxygen. She had continuing congestion in the lungs, noisy breathing. She was not a particularly well baby.

At page 42 of the chart, sir, is the final nursing note which is that of Nurse Nelles, which

you will remember Miss Nelles spent very little time with Baby Miller on the night of March 20th, from the time that Justin Cook was admitted about 10:30. Nevertheless she does record what as we now know was relayed to her and confirmed to her by Mrs. Trayner: her monitor went off several times in the early hours of the morning, the heart rate was slow.

At 1:45 in the morning there was gagging and vomiting. Dr. Soulioti examined the baby and gave her 6 milligrams of lasix by IV push at 2:40.

You will also remember and it appears from page 43 of the chart that at 2:30 Dr. Soulioti wrote an order to hold digoxin. We didn't hear from Dr. Soulioti here. She is no longer in the country I understand.

In Volume 18, pages 3211 to 12 Dr. Rowe agreed that one could reasonably infer from that order written at that time that Dr. Soulioti was concerned that digoxin might be causing the symptoms that she was seeing: bradycardia and vomiting.

Nevertheless the order to hold digoxin was written at 2:30. The lasix was administered at 2:40, and five minutes later Allana Miller demonstrated seizure activity. Her heart rate stopped, a code 25

was called and at 3:27 she was pronounced dead.

At page 41 of the chart is the arrest note. In the lower half of the page, 25 called to 4A/B. Halfway through that note extreme bradycardia initially, complete asystole.

Now you will remember, sir, that blood was drawn at the autopsy of this child at the suggestion of Dr. Costigan, and the digoxin assay which was performed that evening at the insistence of Dr. Carver produced a level of 78 nanograms per millilitre.

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A sample of serum was also assayed at the Centre for Forensic Sciences and the level by RIA there was measured at 69 nanograms per millilitre. That is on page 5 of Exhibit 95A, sir, specimen T29.

A second serum sample was also assayed at the Centre for Forensic Sciences. That is in Exhibit 95B on the first page. Digoxin was identified but is not quantified in that report.

It also appears from 95A, sir, on the fifth page, specimen T10 fixed tissue specimens were assayed at the Centre for Forensic Sciences, and very low concentrations of digoxin were measured in heart, ranging between 5 and 7 nanograms per gram, and in lung tissue 4 nanograms per gram; in each case by RIA only. And the toxologic and pharmacologic curiosity on Baby Miller then is the contrast between the very high serum concentrations and the very low concentrations in fixed tissue.

Now the opinion evidence as to the cause of this child's death is somewhat varied. In the first place Dr. Rowe and Dr. Fowler - you remember Dr. Fowler was ward chief in March - Drs. Rowe and Fowler thought the baby's death was consistent with her clinical condition, as did the CDC's consultant, Dr. Nadas, and Drs. Bain and Taylor.

Interestingly, however, Dr. Freedom to whom the child had been referred and who had followed her course - I seem to have drawn a blank. The only volume out of this huge pile that I need - Dr. Freedom gave evidence at the preliminary enquiry about the child. At Volume 21 of the preliminary enquiry transcript, page 29, lines 9 to 17 he said this:

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Lines 9 to 17 he said this:

"Q. Before you left the baby or at the time you left the baby on the Friday night, well, you've described the surgery that you thought she would require, was her death expected so far as you were concerned?

A. No.

Q. Why do you say that?

A. I felt the child had improved. The rhythm disturbance, while present, was less erratic and the baby looked considerably more comfortable than earlier Friday morning. "

Dr. Fowler was the ward chief and Dr. Rowe was the chief of the division and Dr. Fowler was the person to whom this child had been referred. He saw her the night that she died before he went home. He at least does not seem to have expected her to die when she did.

As to the consistency of the baby's death and terminal symptoms with digoxin intoxication, the pronounced bradycardia and vomiting that were noticed is certainly one of the symptoms of digoxin toxicity that we have learned to recognize.

Dr. Freedom had not attributed her

earlier rhythm irregularities to digoxin toxicity and her heart rate had been slow, as well as irregular throughout her short hospital stay,but with the knowledge of the very high serum digoxin level found in that child, Dr. Freedom, knowing of her earlier irregularities, knowing of her very slow heart rate, considered the death and the circumstances of the death, suggested digoxin toxicity as the cause of death.

That, sir, is found at Volume 29 of our transcript, page 5528.

True enough, it is that at the time that he formed that opinion, Dr. Freedom, like the other cardiologists in the Hospital were pre-occupied I think is not too strong a way to put it, with the digoxin results that they had seen and with what they might mean. At the time they gave evidence here they were a good deal more questioning about the significance of those levels.

Dr. Hastreiter at the preliminary hearing at Volume 34, page 96, gave his opinion that the death of Allana Miller was consistent with digoxin intoxication, a view that was shared by the CDC's consultant. Dr. Kauffman gave evidence here in Volume 71 to the same effect, as did Dr. Mirkin and Dr. MacLeod. Dr. MacLeod's

evidence is of some interest and it is found at Volume 63, page 4204 beginning at line 7:

"Q. At 1:45..."

That is the heart rate of Allana Miller.

"... it drops even lower, it goes down to 54. There is some stimulation. It gets up to 70, drops back again three or four times, we are told.

Then there is vomiting; then there is some seizure activity and then there is an arrest.

Does that sequence of events, doctor, from 1:45, suggest digoxin toxicity?

A. Not particularly to me.

Q. You have a slowing heart rate and increased irregularity, vomiting and some seizure activity?

A. I think it is certainly compatible with -- there is nothing there though that could be said to cry out this is digoxin toxicity.

Q. Is that because it is not so startling a departure from what has preceded it?

A. There really -- it looks like a

"gradual process in fact and bradycardia in fact is not unusual, slowing of heart rate is not unusual in infants as a mode of cardiac death, in my understanding.

The question of vomiting, vomiting stimulates vagal nerve activity and tends to slow heart rate further and might in fact even be a precipitating factor I suppose in the arrest. So there is nothing here that seems strikingly unusual."

As I understand it, Dr. MacLeod, although saying yes those things are compatible with digoxin intoxication, seemed to regard them more as merely a continual of the pattern that had already existed. In fact, Mr. Commissioner, if one looks at the chart at page 24 -- I am sorry, 42. The note as to what occurred at 1:45, appears to suggest a departure from what had happened before, noted to be 54 as though that was something different. Concerning what happened at 2:45 it was regarded, at least by the author of the death notice or death report on this matter, as being a sudden drop out. That is found at page 71 of the chart, sir.

The final paragraph on page 71 of the discharge or death report. And this is Dr. Schaffer's note.

> "The child was started on antibiotics while awaiting culture reports from the septic work. During her stay in the Hospital she was noted to have an irregular heart rate with rates dropping as low as 50 to 60. However, the child always maintained adequate perfusion and B.P. In the early morning of March 21st, the child began developing brady arrhythmias to levels in the lower 50's, however, again with adequate cardiac output. Suddenly, at approximately 2:30 a.m. the child simultaneously became bradycardiac, had a generalized seizure and then became hypertensive. Cardiac arrest was called..."

Certainly there seems to be some room to describe the the events of the early hours of Saturday morning with Allana Miller, as a sudden departure from what had preceded it.

In any event, there is a substantial

body of medical opinion, sir, that Allana Miller's death was consistent, both with her clinical condition and with digoxin intoxication.

The question, therefore, becomes what was the cause of her death. The pathologists, Drs. Cutz and Taylor , the report is found on page 52, assigned digoxin toxicity as the cause of death.

Dr. Rowe did the same thing at Volume 18 of our proceedings, page 3232 and I have already referred to Dr. Freedom, but again fairly I say that the cardiologist's opinions were formed on the basis of serum digoxin concentrations alone.

Dr. Hastreiter, both at the preliminary hearing and here, considered digoxin toxicity the cause of death. Dr. Kauffman certainly was of the view that digoxin had at least contributed to the death. Dr. MacLeod, in Volume 63, beginning at page 4201, gave his view that the likely cause of death was digoxin toxicity. Dr. Spielberg, although he agreed that digoxin toxicity was possibly the cause of death, and he was concerned about it, and said it was not necessarily so.

Dr. Kauffman, when he scored this death at the Centre for Disease Control, gave a digoxin score of 4. You will recall that only one

child of all those that were scored had a higher rating than 4 and that was Justin Cook with a 5.

All of those opinions, as to involvement with digoxin toxicity in the death of this child or as the cause of the death of this child, all those opinions are expressed, notwithstanding the low levels in the fixed tissues. We know from Mr. Cimbura's studies, and, indeed, from Justin Cook's toxicological data that digoxin concentrations in tissue may drop very dramatically, indeed, after the tissue has been fixed in, for example, a Klotz solution.

The significant fact here, in my submission, is that the serum concentration and the terminal symptoms support the views of those who thought digoxin toxicity to be a contributing factor or the cause of the death. It was an unexpected death, as Dr. Freedom said, on the basis of his visit to the child the night that she died.

In my submission, the evidence and the weight of the expert opinion points to digoxin toxicity as the cause of that death. If that be so the question then becomes how much digoxin did she receive, when did she receive it and how was it administered.

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Now you will remember, sir, that one explanation other than administration of a dose of digoxin, one explanation was offered for the elevated serum digoxin level and that by Dr. Spielberg, in Volume 56 at pages 244 - 245 where he raised the possibility of what he called resuscitation trauma. Damage to the heart and vessels during the resuscitation effort causing in some way the dislodging or unbinding of digoxin into the circulatory system, and as he correctly pointed out the proportion of digoxin in the body which is bound is vastly greater than that which is in the circulation of the blood and you don't require to unbind very much in order to raise materially the concentration in the blood. He raised that as a possible explanation of the elevated serum concentrations.

Certainly the autopsy report discloses that such damage did occur to Baby Miller's heart, but it has to be said that there is no evidence other than the fact of damage, but there is no evidence to support Dr. Spielberg's hypothesis. Dr. MacLeod would say only that the suggestion was, and I will quote his words at Volume 63, page 4202:

" Within the realm of possibility. "

Dr. Kauffman thought it unlikely that

resuscitation trauma could produce the post mortem serum levels recorded in this case. I am obliged to make the submission, sir, that dealing as we must in probabilities the overwhelming probability is that Baby Miller's post mortem serum level resulted from an administration of the drug to her during life.

As to the size of dose and time and route of administration Ms. Cronk again has summarized the several opinions that have been expressed by the experts. Only Drs. Kauffman and Hastreiter addressed in any detail the questions. They agreed as to the minimum dose which would be required to produce the recorded level, some where in the order of .5 to .6 milligrams, that is to say one adult vial more or less if given by IV bolus injection. They disagreed as to the likely time of administration. Indeed we have a third and perhaps even a fourth view on the question of the time of administration from Drs. Spielberg and MacLeod. The range is from, as I understand it, about 90 minutes prior to onset of critical symptoms right down to the time of arrest. Dr. MacLeod's time of arrest, as Dr. Spielberg suggested, it could well have been administered then. Dr. MacLeod said administration at 2:40 was quite

possible. Now the 2:40 and 2:45 times from Drs. MacLeod and Spielberg respectively, on the one hand raises the possibility of medication error as being responsible for the administration of digoxin. That is to say the error either at 2:40 in the morning when it was believed that lasix was administered, 6 milligrams by IV push, or the 2:45 time presumably during the course of the arrest proceedings.

Now with respect to those two possibilities of medication error, being the occasion of the administration of digoxin to this child, it first has to be said that there is no evidence whatsoever to support a suggestion that a medication error occurred. But to contemplate the possibilities in any event, if instead of lasix digoxin was given by IV push at 0230, and it has been described as a relatively unlikely error, it was Dr. Hastreiter's view that the amount of digoxin and the appropriate volume of material would be insufficient to produce the recorded digoxin levels in this child. In essence, as I understand it, Mr. Commissioner, it comes to this: lasix is in 1 cc. or 1 millilitre ampules, each of which contains 10 milligrams of the drug, and therefore to draw up 6 milligrams one would draw up .6 of the contents of the ampule, .6 of a cubic

centimetre.

The adult preparation of digoxin as we know comes in 2 cc. ampules, each ampule containing .5 of a milligram of digoxin or a concentration of .25 milligrams per cubic centimetre. If therefore instead of drawing up .6 of a cc. from an ampule of lasix one drew up .6 of a cc. of an adult ampule of digoxin, one would draw up .6 of .25 milligrams of digoxin, that is to say .15 milligrams of digoxin, which is less than the dose calculated by Drs. Kauffman and Hastreiter as being the minimum needed to produce the serum level recorded in Miller.

Now it might be argued that instead of drawing up .6 of a cc. into the syringe one might draw up .6 of the contents of the ampule, whatever those contents might be. If you are dealing with a digoxin ampule and the adult preparation that would be 2 ccs. and if you draw up .6 of that you have got 1.2 ccs. In the first place that in my submission is extremely unlikely. As I understand it, it is the syringe that is calibrated not the ampule. To try to draw up .6 of the contents of any ampule means essentially you are having to eye-ball the ampule and guessing when you have got .6 of it up, much the more rational thing to do would be to draw into

the calibrated syringe the amount that you require. But even if such a crazy thing did occur the result would be that someone would draw up .6 of 2 ccs. of the digoxin preparation and in terms of the amount of digoxin .6 or .5 milligrams, that is to say .3 milligrams is still below the minimum dose postulated by Drs. Hastreiter and Kauffman.

Therefore, in my submission, we do not need to be concerned about the medication error of lasix at 2:40. As for the suggestion that a medication error may have occurred, dig. instead of some other drug, during the arrest itself, again obviously there is no evidence. But also that suggestion assumes that the fixed tissue levels in the Miller samples represent fresh tissue levels and that there was no distribution of the unprescribed dose to tissue. Let me explain that. If a drug were administered during the course of resuscitation efforts, when as we know circulation is at best impaired, one would not expect to see very much distribution to tissue, which rather suggests that the levels recorded in the fixed tissues were close to the levels existing in the fresh tissues at the time of death, there having been very little distribution. In other words whatever is administered

during arrest essentially stays all in the circulatory system and makes up that 69 nanogram level.

Now certainly the low concentration of digoxin measured in fixed tissues, those concentrations are ambiguous. They could mean either that no distribution to tissue occurred, that is to say death followed very shortly afterwards, but probably not as a result of the administration of digoxin, because we know digoxin has no pharmacological effect until it reaches the binding sites. That may be one interpretation of the low fixed tissue concentrations. Or the fixed tissue concentrations may mean that there was distribution to tissue but the digoxin was leached out or degraded as a result of the immersion of the tissue in fixing solution, because we know imperically that that occurs.

Now in one sense it isas legitimate to assume that a dose was given to Baby Miller so late in her life during the arrest as to have no distribution to tissues at all. It is as legitimate to make that assumption as it is to make the assumption that the dose was given at an earlier enough point in time to permit distribution to tissues. If there was no distribution the dig. was all in the serum then the dig. didn't kill Allana Miller. It was given

at an early enough time to permit distribution to tissues in concentrations which are now - it is now impossible to reconstruct because of the fixing process, if distribution did occur to heart then digoxin may well have been the cause of this child's death. So the fixed tissue levels themselves are no help in resolving that conflict. Either assumption on the face of it and looking only at those levels is a legitimate assumption. The fresh tissue levels may well have been higher but no one can say how much higher.

In my submission the weakness of the late administration no distribution theory propounded by Dr. Spielberg is that it explains only the serum level. The only thing that administration during the course of resuscitation can explain is how you get a level of 69 in the serum of that child. It leaves us without any explanation for the occurrence of an arrest in a child whom Dr. Freedom only a few hours before had considered to be improved and whose death he did not expect. It leaves us without an explanation for the symptoms which were recorded, sudden severe bradycardia, vomiting, which although not specific to digoxin toxicity are well recognized as symptoms of digoxin toxicity. One might reasonably

ask what in this child if not digoxin caused the sudden onset of bradycardia preceded by episodes of vomiting. Of course all of the elements of the well-known pattern were present; onset in the middle of the night; one or more members of the same nursing team; inability to resuscitate; and if my earlier submission be accepted the knowledge from Cook's death that there was someone who was not only capable of giving lethal overdoses and indeed did so to Cook. Taking the picture as a whole and considering all of the circumstances, it is my submission that if digoxin was given to Allana Miller by IV bolus injection, it was given earlier rather than later in the range of estimated times, and not by medication error. The earlier injection explains far more than the blood levels. The later administration theory propounded by Dr. Spielberg and to an extent by Dr. MacLeod explains only the blood levels.

I said in formulating that Mr. Commissioner, if given by IV bolus injection, because of course in light of the evidence of Nurse Bell one has to consider whether digoxin was administered to Baby Miller beginning at about midnight via the buretrol, that is to say by slow IV infusion. Dr. Kauffman has said that route of administration

could indeed fit with the timing of the onset of critical symptoms and the arrest of this baby.

Now we have heard from Nurse Bell what she saw, and when she thinks she saw it. It has to be recognized that her evidence as to the time is unclear and uncertain.

We have heard from Nurse Trayner as to what she was doing at the buretrol of Baby Miller's IV at midnight and at 1:00 a.m. Nurse Bell is clear that she saw Nurse Trayner with a syringe in her hand. If she saw that at 1:00 a.m. Nurse Trayner's explanation resolves the matter. If she saw it at midnight, then even though you cannot name names, it is idle to suggest that the consequences for Nurse Trayner would not be very serious. The question for your purpose I suggest, sir, is whether you may rely on Nurse Bell's evidence and on her having seen at midnight what she described to us in order to assist you to conclude that Baby Miller received an unprescribed and unrecorded administration of some medication.

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And if you can form that conclusion on the basis of that evidence, whether you can infer that that medication was digoxin. In my submission if you come to the first conclusion that the evidence of Nurse Bell justifies your concluding that there was an unprescribed dose of something administered to Baby Miller at midnight, then an inference as to the nature of the medication could probably be drawn by you. But it is also my submission that that very serious conclusion cannot properly be reached on uncertain evidence of the kind that is in question here.

THE COMMISSIONER: I am not sure that it can be reached at all in light of the Court of Appeal's decision.

MR. LAMEK: Well, I don't need to take issue with that, sir, because I don't think it could probably be reached in any event.

But in sum on the Allana Miller case it is my submission on the evidence you have heard you may properly find, and again in my submission you should find, that this child died of digoxin intoxication resulting from an administration of an unprescribed dose of digoxin and that there is no satisfactory evidence to suggest that administration

was anything other than deliberate.

I now turn to the case of Kevin Pacsai.

THE COMMISSIONER: I don't want to be too solicitous, but do you want to take - you can take more breaks than usual today if that will assist your effort.

MR. LAMEK: I will soldier on for a while, sir.

THE COMMISSIONER: All right.

MR. LAMEK: The next time I may need to ask for a dispensation of that kind at a later stage.

Mr. Commissioner, in many ways the death of Kevin Pacsai is a puzzle because if digoxin intoxication didn't cause Kevin Pacsai's death no one really knows what did. That is not to say that digoxin toxicity is a diagnosis of exclusion, but there is no other real contender for cause of death.

The expert medical evidence you have heard is almost entirely to the effect that this child's death was not consistent with his clinical condition. Dr. Rowe certainly couldn't say that they were. Volume 17, page 2949 to 50, bottom of the page:

"Q. Are the terminal events that are

"described in the chart and their onset and course consistent with digoxin intoxication?

A. Yes, I think they are.

Q. Are they consistent with what you understood to be the clinical and anatomical condition of this child?

A. Are they consistent with the clinical and anatomical condition?

Q. Well, is this the course that you would have expected, the course of terminal events that you would have expected in Kevin Pacsai, knowing what you did of his physical and clinical condition?

A. No. We couldn't find any reason."

To the same effect is Dr. Fowler in Volume 32.

Dr. Hastreiter in Volume 80, having reviewed this chart, told us he could find no clinical cause of death determined, and he did not perceive the death to be consistent with the clinical condition of the child whose prognosis was good. In his view Kevin Pacsai's cardiac arrest was unexplained.

Dr. Nadas for the CDC was of the view that Pacsai's condition on admission was good and he had a good prognosis. Dr. Mirkin said that death was unexpected from this child's clinical condition, and if it were not for the dig. findings this would be an unexpected and unexplained death.

Now fairly Dr. Kauffman took a slightly different view. Dr. Kauffman in Volume 71, page 5745 said of Kevin Pacsai:

> "...his terminal event was consistent with digoxin intoxication although it could also have been consistent with terminal arrhythmia based on his underlying heart problem."

Dr. Kauffman said he was not of the view that the death was utterly inconsistent with Pacsai's condition. There was general agreement, however, among all physicians and the pharmacologists that the death was consistent with digoxin intoxication.

That was the view of Drs. Rowe, Fowler, Costigan, Cutz, Kauffman, Hastreiter, Mirkin and Kantak and of the CDC. And we know, of course, that the baby's ante mortem serum digoxin concentration was greater than 10 nanograms per millilitre and his post mortem serum concentration was 25, 26 nanograms.

We also know from Exhibit 95A that levels in fixed tissues were recorded, heart and lung and post mortem blood, and what appears in Exhibit 95E, page 5, to be a fresh lung tissue which for some reason Mr. Cimbura regarded to be inconclusive of digoxin intoxication.

Medical opinions as to the cause of Baby Pacsai's death were varied. Dr. Rowe originally thought the baby had died of digoxin intoxication, but by the time he gave his evidence here he wasn't so sure.

Questions had been raised about the interpretation of levels and Dr. Rowe very properly decided that he should defer to the pharmacologists in those matters.

Dr. Freedom really didn't express an opinion as to the cause of death. Dr. Fowler thought possibly digoxin toxicity. Dr. Cutz in the autopsy report said digoxin toxicity. Dr. Kauffman thought digoxin overdose. Dr. MacLeod thought a 75% chance of digoxin toxicity was the cause of death and Dr. Hastreiter, Dr. Mirkin also agreed with digoxin intoxication.

Dr. Spielberg produced the argument involving pathophysiology, and Dr. Bain raised the

suggestion of transient adrenal insufficiency. There seemed therefore to be three contenders: the pathophysiologic argument of Dr. Spielberg, the transient adrenal insufficiency of Dr. Bain and the digoxin intoxication cause to which most of the medical men who have given evidence here subscribe.

As I understand the pathopsyiologic theory of Dr. Spielberg it really goes to explain the high digoxin level and how that could occur without an administration of digoxin. And the theory - I don't want to misstate it and do violence to the thing - was stated by Dr. Spielberg in Volume 55 beginning at page 2312.

Forgive me, Mr. Commissioner, but I think to do justice to him one must go to Dr. Spielberg's own formulation of it. Beginning at line 7 on page 2312, the question which was one of those - it is called a question; in fact it is the answer I want you to know and I am not responsible for this very long thing. For Q. read A.

> "A. Well I think probably a year ago the only two possible explanations we would have had for Kevin's problem would have been administration of digoxin in some way or another, with an amount calculated to produce his

"level of ten, or perhaps a little bit greater, going up to 26 post mortem. But there have been certain things that have occurred over the last year, both in the published literature, as well as at least one infant who already has been the subject of an inquest, and several other infants who have been followed on the cardiology floors on 4A/B, where blood levels have exhibited what we will call D distribution phenomena, which is one thing that I think we have to accept as one potential explanation of what happened to Kevin. Now under circumstances such as a level of ten, a year ago we might have thought that this could never happen in a living patient, and in fact we have seen it now in several patients, where a level despite no excessive administration of digoxin, for reasons which in some patients in the literature have been explained on the basis of renal failure, and other patients have been explained on the basis of tissue

"damage, to a variety of different organs in a variety of different ways. Unfortunately with so many variables that we can't always define them, but patients whose serum digoxin levels have continued to rise to levels in fact very much in the range of ten during life despite no administration of digoxin.

Now the question - that does not mean they didn't have digoxin in the first place, this is presupposing that the infants indeed had digoxin in their bodies but that a small fraction was lost during life. What does this mean in terms of Keven Pacsai? The enigma we face is the fact he re-established sinus rhythm in the face of an apparently elevated digoxin level and the rhythm disturbance, and in fact lived four hours."

There was then an exchange with which I don't need to bother you, sir. Going to page 2314, line 18, Dr. Spielberg said:

"Now struggling with those numbers,

"I don't have a good explanation. I believe the possibility still exists that the child could have been given an excessive dose of digoxin, much smaller quantities of course than these other babies, because the levels are much lower ...

I think the other possibility has to be considered, that in light of other babies that we have now seen, as well as the published literature that increases in serum digoxin level from tissue loss may have occurred in this baby, thus the baby's serum concentration would have been increased, but the concentration at his myocardium might not have been increased, and in fact might have been slightly decreased. Because again to go to the level of 10 or 20 from a level of 1.8 is a very tiny fraction of loss of digoxin as we discussed yesterday. We are not talking about massive digoxin release, we are talking about probably two per cent, maybe three per cent,

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"a very, very small amount of release, from mechanisms that again in honesty we don't understand, except that we have seen it in other patients.

Thus we have a situation where the baby's total body digoxin was the same, but where his serum level in fact was higher. Under those circumstances I find it easier to imagine the child going back into sinus rhythm. The fact that he then reverted to an abnormal rhythm is basically what had been happening to the child all along. In fact the child had tremendous rhythm disturbance and was going up and down and up and down, and that he finally died from a rhythm disturbance is not surprising.

Thus I think the three possibilities that exist in the infant, that I have to at least consider pharmacologically is: one, accidental or intentional administration of digoxin..."

He meant those to be two -

"...and two, abnormal pathophysiology

"with a rising serum digoxin level as a result of phenomena that again we do not understand, but that in fact we see."

Dr. Spielberg is postulating that by some means or mechanism digoxin already in the child in some way becomes unbound, goes into the circulatory system, manifests itself as an elevated level there, but obviously without having any enhanced pharmacological effect on the child (if anything having a reduced pharmacological effect) and that he says may account for the high digoxin level and it might also account for the return to sinus rhythm that was seen in the case of Kevin Pacsai.

So there are two possible explanations according to Dr. Spielberg for Pacsai's high digoxin levels: one, he received a dose of digoxin, large, not enormous, or two, some pathophysiologic process was at work in his body causing to some measure the unbinding of digoxin from the tissue.

Now that pathophysiologic theory had been advanced by Dr. Kauffman in the case of Gary Murphy in 1983 as you will remember, sir.

Dr. Kauffman's response to Dr. Spielberg's attempts to apply that approach to Kevin Pacsai

is found in Dr. Kauffman's evidence here in Volume 72. It begins at page 5808 and goes on, frankly, to 5825 and I don't propose to read it. I don't think I could. But I think I summarize it fairly when I say Dr. Kauffman said no, I can't accept --

MS. CRONK: The exchange was a little bit better than that.

MR. LAMEK: When I summarize I summarize.

MS. CRONK: I am sorry, sir. I couldn't resist.

MR. LAMEK: He said the cases of Gary Murphy and Kevin Pacsai are not analogous. He did what lawyers do: he distinguished the two. He said that Gary Murphy's physical condition and his clinical condition were so much worse than those of Kevin Pacsai that what may have been a reasonable hypothesis in his case just does not fit the Pacsai case in Kauffman's view.

The core of the Kauffman response to the pathophysiology theory as applied to Kevin Pacsai is found, sir, at page 5810 beginning at line 13 to 5811 at line 20 and 5822, line 2 to 5825, line 7, all in Volume 72.

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It is my submission, sir, that Dr. Kauffman appears to me at least to have satisfactorly distinguished the two cases and has shown why in his view Dr. Spielberg's proferred explanation of Pacsai's high digoxin levels is invalid. I mean no offense to Dr. Spielberg. He was clearly making an honest attempt to canvass all explanations for the observed phenomena, but it is my submission that Dr. Kauffman's position makes practical sense and that the case and condition of Kevin Pacsai are not so unusual that one needs to look for hypothetical and speculative explanations for an elevated serum digoxin level when a much more natural explanation lies ready to hand, that is to say that he received an unprescribed dose of the drug.

Now, as for Dr. Bain's theory that Kevin Pacsai suffered from and died of transient adrenal insufficiency, there is really little to say. It is a theory which, by the nature of the diseased condition which it postulates, can be neither proved nor disproved.

The catch 22 about transient adrenal insufficiency is that you can't detect it, because it is transient. It comes and does its deadly thing and it departs without a trace. I don't intend to derive the theory, but with the utmost respect to Dr. Bain, it can be no more than a theory.

As Mr. Shinehoft pointed out, the theory postulates a disease condition which is virtually unknown in the literature and whose visitation can't be detected at autopsy. Dr. Cutz said that as well, at Volume 42, page 8578.

In this case, according to Dr. Cutz, the pathologist, Kevin Pacsai's adrenal glands were grossly and microscopically normal.

Dr. Bain's suggested diagnosis was properly treated, respectfully by Dr. Rowe and the other physicians. I would not wish to appear to treat it disrespectfully, but I am obliged to say that it is difficult, in my position, to accept an unprovable diagnosis when another candidate apparently far more plausible presents itself. The candidacy of digoxin intoxication to be accepted as the cause of death of Kevin Pacsai is based primarily on two matters. The first is recorded ante mortem and post mortem serum levels and, second, is terminal symptoms.

In respect to the terminal symptoms, page 63 of the charts sets them out. I don't need to ask you to turn to them, sir. They exhibited sinus bradycardia, sinus or nodal tachycardia, intermittant 2 to 1 heart block. Symptoms more severe than he had hitherto shown. There were symptoms which Dr.

Costigan recognized as among those associated with digoxin toxicity which he noted as a differential diagnosis and he ordered digoxin held.

Dr. Costigan's note in the I.C.U. is contained at page 66 of the chart. It records that on leaving the ward Pacsai of bradycardia down to 40. Although there were further episodes of bradycardia with 3 to 1 heart block. He recorded his impression as brady arrhythmia second rate to digoxin toxicity or sinoartrial nerve disease. It is true that Baby Pacsai had a history of arrhythmias. He was sent to the Hospital for Sick Children partly for investigation in his bradycardia. Dr. Costigan knew that. He knew what the history was. He records it on the I.C.U. transfer note. He was now seeing something in this child that was apparently sufficiently different from and sufficiently more severe than anything seen earlier as to make him think in terms of interalia digoxin toxicity.

In my submission there is ample evidence and a substantial body of medical opinion to justify, if not absolutely to compel, a conclusion that Kevin Pacsai did indeed die of digoxin intoxication.

I am about to move to how, when and how much, Mr. Commissioner. May I take your offer of a break

now, please.

THE COMMISSIONER: Yes, we will take 20 minutes.

MR. LAMEK: Thank you.

--- Short recess.

--- On resuming

THE COMMISSIONER: We have attempted to work on the invalid, but without any great success and as a result we will cover the Janice Estrella matter, which will probably take us up until the lunchbreak, in any event, and then we will just -- I don't know. We will take a holiday, but he, I trust, will take some of the better known medical -- I don't really care what it is as long as it does the job. I don't know where that leaves you, Mr. Scott, but -- provided of course this illness is not terminal he will finish it on Monday. Does that mean you will or will not be able to proceed on Tuesday?

MR. SCOTT: I will be able to proceed on Tuesday. Much depends on Miss Thomson.

THE COMMISSIONER: I see.

MR. SCOTT: Much depends on what he says in the closing minutes of his oration. I don't seek any delay at the present time. I think we can go on with it.

THE COMMISSIONER: Thank you. Yes, Mr. Lamek.

MR. LAMEK: Miss Cronk reminds me that in making the submission to you that you should find, as the cause of death of Kevin Pacsai, digoxin intoxication, that there is a further matter which goes to form the basis for that conclusion, in addition to those that I mentioned, and that is that the level recorded in the fixed and frozen lung tissues of this child were clearly in that range described as toxic by Mr. Cimbura. We are not only dealing with serum concentrations.

When we get to the question of how and when Kevin Pacsai received the dose of digoxin, which I have suggested you shall find he did receive, and the size of that dose, we come to the point when the puzzles and the mysteries really begin. The difficulties lie in two areas. In the first place, this child seems to have suffered an onset of critical symptoms at approximately 4:00 o'clock in the morning. Those symptoms progessed in a more or less unbroken path when eventual cardiac arrest in the intensive care unit at about 8:50. Resuscitation efforts there were unsuccessful and he was pronounced dead at 10:10. That is to say a very long period elapsed here between

the onset of critical symptoms and the arrest, almost five hours.

Secondly, by contrast with the levels recorded in the other babies that we have looked at, Cook and Miller, perhaps Estrella to come, the Pacsai serum levels, although well into the toxic range, are not astronomically high.

Those two features of the Pacsai matter come together to make questions of the route of administration and the size and the time of the dose very much more difficult for the pharmacologist to form a judgement about and their opinions, as you heard yesterday from Miss Cronk, and as they have been expressed here are widely divergent and frequently diametrically opposed.

Summarizing very briefly, and knowing that Miss Cronk got into them in detail, Dr. Hastreiter changed his view between the time of the preliminary inquiry and the time he gave evidence here. In the preliminary hearing he said that he really couldn't express a competent opinion as to the route of administration, but he thought a slow IV infusion was likely.

Here he said that a slow IV infusion was possible and an IV bolus injection was most likely,

most possible and most probable, was the language that he used on three different occasions. He also told us that oral admission was in his view unlikely.

Dr. Kauffman on the other hand, said he really wasn't sure whether if we were dealing here with oral or IV administrations but he also said that he considered oral administrations most likely, but IV administration was possible.

He then said, at a later point, that IV was most likely an oral administration was possible. He also said he considered it unlikely that the administration was by IV bolus injection shortly before death.

Dr. MacLeod considered oral administration most likely, IV administration unlikely and Drs. Mirkin and Spielberg wisely refrained from expressing any view on the question.

The opinions on size and time of dose are obviously, to some extent, influenced by the route of administration, which the pharmacologist thinks to be most likely and Dr. MacLeod gave his opinion, not withstanding he thought IV administration unlikely, gave his opinion as to the size of an IV dose that would be required. It was about an adult ampule. At the time he said that if that dose were administered

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between 3:30 and 5:30 he would have expected effects to be seen much earlier than they were, in fact, seen. He did say that if the digoxin were administered orally between 3:30 and 5:30 it would be the most likely time.

Dr. Kauffman was very cautious. He said an excessive amount of digoxin was received by Baby Pacsai. He gave the minimum oral dose estimate at .7 milligrams.

Continuing in his rather bold view of things he said the time of administration was hard to assess and he gave certain earliest possible times.

Mr. Commissioner, I see no point of multiplying examples of disagreement among the experts on those questions. There was an area of agreement between Drs. Hastreiter and Mirkin, each of whom said that the earliest possible time for administration whether oral or by IV slow infusion, was 2:00 o'clock in the morning.

In short, the size of the dose of digoxin that most of the pharmacologists believed to have been given to Baby Pacsai and the time and the route of administration of that dose, are matters about which, on the conflicting contradictory evidence you have heard, it may well be impossible to make a finding.

The question is what then is clear about Baby Pacsai? I suggest a number of things are clear. First, his death was unexpected, an opinion that was shared by Drs. Rowe, Fowler, Hastreiter and apparently Nadas and Mirkin.

Second, there is no clear clinical cause of death. Third, he suffered a sudden and severe deterioration. You will recall Miss Nelles' evidence that when she returned from helping at the Manojlovich's arrest, Kevin Pacsai was not the same child that she had dealt with earlier on that shift. That is somewhat reminiscent of Nurse Scott's observation. You will remember that she said that she would go on a break, leaving a comfortable, stable child and within an hour of her return the child would be in serious difficulty and would die. The same sort of thing appears to have happened with Miss Nelles. She left Pacsai to go out and assist in the other arrest that night. When she got back it was a very different child that she got back to.

Pacsai suffered an arrest in the early hours of the morning. He certainly suffered his onset of critical symptoms at an early time, the arrest, some hours later. He suffered that onset of critical symptoms in the presence of members of that same

nursing team.

His deterioration following the onset of critical symptoms although it extend over a period of time, was replete with episodes of symptoms associated, although not exclusively associated with digoxin toxicity, severe bradycardia, heart block and ventricular fibrillation.

It is also clear that he had an ante mortem and a post mortem serum digoxin concentration in the range normally regarded as indicative of severe, if not fatal, intoxication. He had tissue concentrations described in the same way.

It is also clear that he had a structurally normal heart quite what the significance of that is in terms of his decline in death, is not at all clear.

We have heard different opinions on the one hand. It may have enabled him to withstand the effects of digoxin for a longer period than would have been possible had his heart been defective. On the other hand although he did have a rhythm disturbance problem, it may have worked the other way to make him more susceptible to the effects of excessive digoxin.

If Baby Pacsai did receive an unprescribed and excessive dose prescribed, there would be no suggestion on the evidence, which I can recall or

which I have been able to find, that there was any medication error involved.

.....

I suppose the possibility always exists, but there was no particular incident to which anyone has pointed that there was an occasion upon which medication error could have occurred.

In all the circumstances, sir, although you may not feel able to express any conclusion about how or when digoxin was given to Baby Pacsai, or how much was given, you would in my submission be justified in finding the child died of digoxin toxicity resulting from the administration to him of an unprescribed and excessive dose of digoxin, and that there is nothing in the evidence to suggest that that administration was accidental.

Now, Mr. Commissioner, the last of the babies in this group, that is to say the babies whose deaths were the subject matter of the charges against Miss Nelles is Janice Estrella. She died at the age of 4 months at 3:22 in the morning in room 423, ward 4A, on January 11th, 1981. She had had surgery on December 16th but she was not doing well. The virtually unanimous view of the physicians was that her death was entirely consistent with her clinical condition. That was Dr. Rowe's view, that was shared by Drs. Fowler, Nadas, for the CDC, Bain, Kauffman and Hastreiter. The only slight dissent was

from Dr. Mirkin. He thought her condition to be stable and her heart rate and rhythm normal and he therefore considered her death to be somewhat unexpected. And fairly, although Dr. Hastreiter gave Janice Estrella a severity rate of 8 out of 10 and said she was expected to die, he too considered that she was not expected to die just when she did.

The vast preponderance of the medical evidence has been that Janice Estrella's death was not surprising in light of her clinical condition.

On the night of her death Baby Estrella was under constant nursing care, and that meant of course that a nurse had to be with her at all times. You will recall that we heard evidence from Nurse Scott, the nurse that was assigned to provide constant nursing care that night, and from others, that could lead you to conclude that the baby was left alone for a few minutes at about 2:00 a.m.

Now in the 24 hours preceding her death, and this no doubt is the matter upon which Drs. Hastreiter and Mirkin relied for their inference that perhaps death wasn't expected just when it occurred; in the 24 hours preceding her death Janice Estrella had appeared to be relatively stable. On

page 125 of the chart is the long night nursing note for the night preceding her death and Sui Scott had provided constant care that night also. The heart rate was regular all night. Fast breathing all night, the other vital signs were stable. Tolerating formula well and fed by NG tube. Voiding well, she was pale, she was in oxygen and slightly clammy.

On January 10th Dr. Tucker wrote a note, the temperature was elevated, although she was quick to point out the sun was shining through the window onto the crib. It is hard to think that January sun would have made that much difference but no doubt Dr. Tucker thought so. The chest sounds clear she said, respiration a little fast, the pulse is up slightly no signs of congestive heart failure.

Page 126 the chart the same general picture; apex regular all day in the nursing note, still breathing fast, temperature up during day, colour better, however still is pale.

A further note by Dr. Tucker at 11:30 p.m. on January the 10th. Temperature is down, chest is clear; the child is stable, she says no signs of congestive heart failure.

Page 128 of the chart, Mrs. Scott's nursing note for the long night January 10 to 11 the night the child died. This is the first part of the shift, much the same picture. Apex is regular throughout and strong and so on and so on. Then at 2:40 in the morning a sudden and very dramatic turnaround. Nurse Scott had been back from her break about 30 to 40 minutes at this stage. She finds the baby slightly gasping, respiration rate rapidly dropping, faint heartbeat and a code 25 was called.

Two pages earlier in the chart, this is page 126, Dr. Tucker's arrest note:

" Baby found bradycardic; respiratory rate down; cardiac arrest team called. "

It goes on:

" No response child pronounced dead at 3:22. "

Dr. Tucker described this child as stable a little over three hours before her sudden death. Nurse Scott had left an apparently stable child to go for her lunch at about 1:30 and before 3:30 the child is dead. Now again not a well child, indeed a seriously sick child, but once again a child

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who in the middle of the night and in the presence of the same nursing team has gone from relative stability into an irreversible decline almost in the blinking of an eye.

For a more complete statement of the terminal symptoms exhibited by Janice Estrella, I refer you sir, to page 33 of the chart which is the death report. Beginning eight or nine lines from the bottom in the middle of the line:

> "In the early morning of January the 11th, at approximately 2:45, after the child had been previously noted to be in sinus rhythm, the child suddenly developed slow breathing and bradycardia and a cardiac arrest occurred. At the time of arrival of the Cardiac Arrest Team, the child was noted to be in ventricular fibrulation which then converted spontaneously to a slow supraventricular bradycardia. Despite maximum medical intravention, the child was unable to be resuscitated and was pronounced dead at 3:25 a.m."

So you have got the arrhythmias that we have seen in so many of these children in the course of the

evidence, bradycardia to ventribular fibrulation, tachybradycardia to death.

I have referred already to the evidence relating to the ordering of a post mortem digoxin level on the Baby Estrella, that was a matter to which I referred on the opening day of this week. No matter how that order came about, and no matter whether the Dr. Freedom can recall it or not, no matter what he believed he was doing at the time, samples were drawn in the way about which we have heard some much and it produced readings of 72 nanograms per millilitre in pelvic cavity samples and greater than 4.7 nanograms per millilitre in the leg vein sample.

The generally held view among the physicians from whom we have heard is that their views of the cause of death of Janice Estrella depend on the validity of that 72 nanogram level in the sample drawn from the pelvic cavity. If that level is reliable, there is a substantial body of opinion that says the probable cause of death is digoxin toxicity. If it is not, the probable cause of death was the child's clinical condition. Dr. Kauffman's was typical of such a response. His original opinion was that Baby Estrella's digoxin level was excessively high and was

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compatible with a lethal overdose. When he learned that the 72 nanogram level was measured not in venus blood as he had thought, but in blood from the pelvic cavity; and when he learned the results of the so called gutter blood study he felt that he had to revise his original opinion substantually. Now although he says he would not dismiss the 72 nanogram level completely he has to have very much less confidence and he now finds it very difficult to form a conclusion about digoxin toxicity in Janice Estrella.

Dr. Hastreiter too felt the case for digoxin overdose was considerably weakened by the results of the gutter blood study. That study is Exhibit 238, sir, and I don't necessarily ask you to turn to it now. You will recall that with one sample drawn from the pelvic cavity is grossly out of line with other samples from the same child.

When Dr. Mirkin reviewed all the results of the gutter blood study and observed that only one sample out of, I think it was 24, 25, or 26 and that was a matter of some debate produced a distorted level he said this, and it is found, sir, in Volume 88, page 9034 to 9036. In the course of cross-examination I believe by Mr. Brown, beginning at line 10 he had had the gutter blood study results

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put before him and he said:

"Reading these data certainly makes me question whether the lack of confidence in those data in the 72 value is warranted I think that indeed may be an accurate reflection of what was there; let's make that a point. Because now one would have to reconsider why one would call that particular value unreliable in the face of the study that you have just presented which suggests that the gutter levels whether taken at the post mortem or three hours thereafter are really a very accurate reflection of what is in the heart blood or the sagittal sinus, or the heart blood let us say."

That was a small exchange, and he goes on :

"The point I am making is that these data suggest that the gutter blood may actually be an accurate reflection of what is in the systematic blood of the patient. Okay, is that clear?

Q. Yes.

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A. I think it is important though for you to understand that our evaluation at the time I made it also made that presumption that the 72 was a valid number.

Q. Yes.

A. Okay. So now one can go back to accepting those data with more confidence as a consequence of these data."

Dr. Mirkin seemed to find a comfort, not distress in the results of the gutter blood study.

In Volume 89, at page 168 he said this and this is in the course of re-examination by me:

"Mr. Brown yesterday referred you to the results of what we call the gutter blood study in the context of a discussion of Janice Estrella and that child's death. In particular he showed you the particular results of that study and I think you will agree with me that although you have not seen the particular results, the substance of the results has been outlined to you prior to your

"giving evidence.

As I understood you, you said that the reasonably good correlation between all but one of the results reported in gutter samples and the levels reported in heart blood from the same children enable you to have a reasonably good confidence level in the 72 nanograms level reported in Estrella, not withstanding that there was one extremely high and anomalous gutter blood level in the study. That is a rather tortuous way of summarizing your evidence, but do I put it fairly?"

He agreed that I did:

"Q. In short, the existence of the one anomalously high reading of the gutter blood study does not greatly shake the confidence that you are prepared to place in the post mortem Estrella's sample.

A. Correct.

Q. And as I therefore understood it you are prepared to revise again, or rerevise your opinion as to the probable

involvement of digoxin in the death of Janice Estrella.

A. Based on this post mortem information.

Q. And do I understand it that you are now inclined to think that digoxin intoxication did indeed play a part, if it was not indeed the cause of Janice Estrella's death.

A. I think that is correct. "

I then put to Dr. Mirkin the caution that Drs. Kauffman and Hastreiter had expressed in light of the gutter blood study and I will attempt to summarize this. Dr. Mirkin said, all right, what we have got here is 25 out or 26 samples which indicate that gutter blood is a perfectly proper reflection of blood from other parts of the body. He regarded that as statistically comforting in enabling him to rely upon the 72 level taken in blood from the gutter in the case of Janice Estrella.

There, Mr. Commissioner, you have the competing views as to the effects of the gutter blood study on the reliability of the 72 level from Estrella. Does one cease to rely on that 72 nanogram levels because once in 26 times such samples can produce falsely high levels, or does one feel greater confidence

because 25 times out of 26 those samples produce reliable results. Rather like asking whether the glass is half empty of is it half full.

It is my submission that in all the circumstances of this case Dr. Mirkin's view is to be preferred and I say that for three reasons. First, as you have repeatedly said over the course of the past year, this is not a murder trial, and that is even more clearly so since the Judgement of the Court of Appeal no one thinks it is.

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You will not I know make any finding, however, in this matter lightly or on flimsy evidence, but you do not need to be convinced beyond a reasonable doubt before you find something to be thus and so. No doubt if one were searching for absolute scientific proof, one would tend to be extremely conservative in one's approach to this Estrella post mortem serum dig. level. I suggest this is not necessarily that kind of exercise.

I say respectfully, sir, you are required to bring your best judgment to bear on the matter before you and to do your best to answer the questions that have been troubling so many people so long,and in my respectful submission --

THE COMMISSIONER: I am not like a jury; I do not have to say one or the other.

MR. LAMKE: That's right, you don't have to say one or the other.

My point is merely this, sir: you do not have to say beyond any question of scientific validity this sample is or is not a valid one. But in saying that to you and making it too in the context of other considerations one of which is these: to reject the 72 nanogram level is to ignore the other indicia of digoxin intoxication in this case which

although not compelling in themselves are some added evidence of digoxin toxicity.

I refer, of course, to the terminal symptoms exhibited by this child. They are symptoms which presumably were considered consistent with digoxin toxicity by all of those physicians who reviewed this case and said, yes, death is consistent with that cause of death.

They are still in my submission matters that may be taken as corroborative to some degree of digoxin toxicity, and to that extent may lend some support to the 72 nanogram levels.

And third, of course, there are the non-medical circumstancial evidentiary matters that cannot be ignored: the patterns, the common threads. They are all present in this case.

In short, Mr. Commissioner, what we have in the case of Janice Estrella is a package made up of all of the common threads, terminal symptoms that are suggestive of digoxin toxicity and a grossly elevated serum digoxin level in a sample which may be unreliable but which as a matter of statistical probability is likely not to be unreliable in the judgment of Dr. Mirkin.

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In light of the whole picture it is my submission you may properly find that Janice Estrella too died of digoxin intoxication resulting from the administration to her of an unprescribed and excessive dose of digoxin.

Now, Mr. Commissioner, those are the four children with whose murder Miss Nelles was charged. My submission is that the evidence justifies the conclusion that each one of those children either clearly or with a very high degree of probability died of digoxin intoxication as a result of an administration of an overdose of that drug and that there is no basis on the evidence for concluding that any one of those adminstrations was anything other than deliberate.

Before I leave that group of four, Mr. Commissioner, may I just say this: I recognize fully that Dr. Rowe and others have told us that a sudden and rapid decline from a period of apparent stability is no necessary indication of intoxication and that any and all young cardiac patients could follow such a terminal course.

I want to explore that a little more latter, but for the moment I do observe that of the children for whom I have made submissions so far, three

of them who in my submission did die of digoxin intoxication did follow a very similar terminal course. A course that was sudden in onset and rapid and irreversible in its program.

The fourth, Pacsai, certainly had a sudden decline. It was no less irreversible, but admittedly was less rapid than those of the other three. Whether the distinction be attributable to the administration of a smaller overdose, to a different route of administration, to the fact that his heart was structurally normal, or a combination of those and other factors, I do not know, but even with Pacsai one thing is clear in my submission: not one of these four children just drifted quietly away to the point of death.

Mr. Commissioner, I propose to move next to those three other children, three other than Cook, for whom digoxin was not prescribed and in whose bodies it was found and I would be most grateful if I could do that on Monday morning.

THE COMMISSIONER: Yes.

MR. LAMEK: Thank you.

THE COMMISSIONER: All right. Unless anybody has anything else we will rise until 10:00 o'clock on Monday morning.

---Whereupon the hearing was adjourned at 12:15 p.m. until 10:00 a.m., Monday, June 11th, 1984.